Pulmonary Arterial Hypertension Related to HIV Infection: Improved Hemodynamics and Survival Associated with Antiretroviral Therapy

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This study aimed to assess the long-term course of pulmonary arterial hypertension related to infection with human immunodeficiency virus (PAHRH) and the influence of antiretroviral therapy (ART) on its characteristics. We retrospectively analyzed all 47 patients in the Swiss HIV Cohort Study in whom PAHRH was diagnosed. Among 35 patients who underwent follow-up Doppler echocardiography, the right ventricular systolic pressure over right atrial pressure gradient increased by a median of 25 mm Hg in 9 patients who had not received ART, decreased by a median of 3 mm Hg in 12 patients who had received nucleoside analogs, and decreased by a median of 21 mm Hg in 14 patients who had received highly active ART (HAART) ($P < .005$). Among all 47 patients, median duration of survival after PAHRH diagnosis was 2.7 years. HAART significantly decreased mortality due to PAHRH as well as other causes. This study suggests a beneficial effect of combination ART in patients with PAHRH.

The association between pulmonary arterial hypertension (PAH) and HIV infection is well established [1–7]. It was first described in 1987 in an HIV-infected patient with hemophilia and membranoproliferative glomerulonephritis [8]. To date, ~220 cases of PAH related to infection with HIV (PAHRH) have been reported. The incidence of PAHRH is estimated to be ~0.5% [2, 3], which is ~2500 times greater than that of primary pulmonary hypertension (PPH) in the general population [9]. The pathogenesis of PAHRH remains unclear. There is no evidence that HIV infects pulmonary artery endothelial cells. Neither HIV nor its proteins has been identified in the pulmonary vascular endothelium of patients with PAHRH [10]. It has been suggested that the increased incidence of PAH in patients with HIV might be the result of an indirect role of the virus, stimulating the host to release proinflammatory cytokines or growth factors that would result in PAH [11–14]. Only a small percentage of HIV-infected patients, however, develop PAH. This supports the existence of an individual susceptibility to the development of this disease. A recent study suggests that this susceptibility could have a genetic basis and might be determined by major histocompatibility complex alleles, particularly *HLA-DR6* and *HLA-DR52* [15], but preliminary unpublished examinations suggest that these alleles are not present in HIV-infected patients with PAH. On the other hand, the histopathology of HIV-associated pulmonary vasculopathy is similar to that of PPH [1, 2, 6, 10, 16, 17], suggesting similar etiological mechanisms. This is supported by data showing that PAHRH also responds to treatment with the endothelin-receptor antagonist bosentan, which is effective in patients with PPH [18, 19].

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The records of the Swiss HIV Cohort Study (SHCS) [20] contain one of the largest cohorts with PAHRH. In this study, we retrospectively evaluated all patients with PAHRH in the SHCS. One goal was to describe the long-term clinical and echocardiographic course of the disease.

The best therapeutic approach for patients with PAHRH is unknown [21, 22]. Few reports describe the efficacy of treatment for PAHRH. Drugs used in the treatment of PAHRH include anticoagulation therapy [22, 23], diuretics [22], vasodilators (calcium channel blockers [24, 25], intravenous epoprostenol [1, 26, 27], inhaled iloprost [28], sildenafil [29, 30], and bosentan [18]), and antiretroviral treatment (ART) [24, 27, 31–33]. In a previous study, Opravil et al. [24] suggested that ART decreases the right ventricular systolic pressure (RVSP) over right atrial pressure (RAP) gradient in patients receiving ART, compared with an increased gradient in patients who were not receiving ART. On the basis of improvements in the pressure gradient over time in patients who received ART, it was recommended that all patients be treated with PAHRH, irrespective of their CD4 lymphocyte counts. On the other hand, an accelerated course of the disease has been reported in 2 patients who received HAART [31]. Thus, another goal of the present study was to evaluate a larger number of patients over a longer follow-up period to determine whether the pressure gradient could be influenced by ART and whether mortality attributable to PAHRH could be decreased.

PATIENTS AND METHODS

Patients. We retrospectively evaluated all 47 patients who received a diagnosis of PAHRH between 1988 and December 2001 and who were observed within the SHCS. Some patients were examined prospectively with successive echocardiography. Twenty-nine of the cases we describe have not been reported previously. Diagnostic criteria for PAHRH were as follows: elevation of RVSP over RAP of >30 mm Hg, assessed by continuous-wave Doppler echocardiography; exclusion of pulmonary emboli by lung perfusion scan or autopsy; normal left ventricular function and no evidence of congenital or valvular heart disease (evaluated by 2-dimensional echocardiography); exclusion of obstructive or restrictive pulmonary disease on the basis of clinical examination and pulmonary function tests; and no history of previous ingestion of anorectics or use of intravenous amphetamine therapy. The diagnosis of PAH was invasively confirmed in 8 patients who underwent right heart catheterization. The catheterization values were in good concordance with echocardiographic findings. To assess the possible effect of ART on the course of PAHRH, patients were divided into 3 groups on the basis of the treatment received between the time of PAHRH diagnosis and the performance of the final echocardiography (or the final clinical follow-up, for those who did not undergo successive echocardiography): those who received <4 weeks of ART or no ART at all, those who were treated with 1 or 2 nucleoside analog reverse-transcriptase inhibitors (NRTIs) only, and those who received HAART, which was defined as triple therapy including at least 1 protease inhibitor or non-NRTI.

Statistical analysis. Comparisons between the 3 treatment groups were evaluated using χ^2 analysis or Kruskal-Wallis testing with Dunn's multiple comparison posttest, as appropriate. Predictors for decreases in the pressure gradient were assessed by multiple logistic regression analysis. The risk of death and predictors for survival were calculated by means of Kaplan-Meier life-table methods and multivariable Cox regression analysis. Analyses were conducted using GraphPad Prism, version 4.00 for Windows (GraphPad Software), and Stata software, version 8.2 (Stata).

RESULTS

Demographic and clinical data. Between January 1988 and December 2001, 47 patients fulfilled our diagnostic criteria for PAHRH (table 1). There were 25 men (53%) and 22 women (47%), with a mean age at the time of diagnosis of 34.4 years (range, 24–67 years). Forty-six patients were white and 1 was black. Injection drug use (IDU) was the most common risk factor for HIV infection and was reported by 33 patients (70%). Other risk factors reported included male-male sex by 7 patients (15%), both heterosexual sex and IDU by 3 patients (6%), and heterosexual sex by 4 patients (9%). The median time interval between the diagnosis of HIV infection and the diagnosis of PAHRH was 7.7 years. At the time of diagnosis of PAHRH, 17 patients were in US Centers for Disease Control and Prevention HIV disease stage A, 18 were in stage B, and 12 were in stage C. The median CD4 lymphocyte count at the time of diagnosis was 169 cells/μL (range, 1–870 cells/μL). Twenty-eight patients had CD4 cell counts of <200 cells/μL, 14 patients had CD4 cell counts of 200–499 cells/μL, and 5 patients had CD4 cell counts of >500 cells/μL. The median New York Heart Association (NYHA) functional class value was 2.

The most common presenting symptom leading to the diagnosis of PAHRH was shortness of breath in 83% of the patients. Other presenting symptoms included peripheral edema in 26% of the patients, syncope in 6%, and chest pain in 6%. An electrocardiogram, which was available for 42 patients, showed signs of PAH in 79%. Findings on chest radiographs were consistent with PAH for 74% of the patients. The mean elevation ($\pm$SD) of RVSP over RAP was 63 $\pm$ 16 mm Hg, ranging from 38 to 115 mm Hg at the time of diagnosis. No patient had stenosis of the pulmonary artery, and no patient had systemic hypertension. The 3 treatment groups did not differ with regard to their baseline characteristics, except that

Table 1. Patient demographic and clinical characteristics at the time of diagnosis of pulmonary arterial hypertension associated with HIV infection.

Characteristic	Overall population (n = 47)	Treatment group: No ART during follow-up (n = 13)	Only NRTIs during follow-up (n = 15)	HAART during follow-up (n = 19)
Male sex[a]	25/47 (53)	6/13 (46)	7/15 (47)	12/19 (63)
Age, years	33 (29–36)	34 (28–36)	31 (28–33)	36 (32–41)
Median date of PAHRH diagnosis	January 1994	May 1993	March 1993	February 1998
HIV transmission category				
IDU	33 (70)	13 (100)	10 (67)	10 (53)
MSM	7 (15)	0 (0)	2 (13)	5 (26)
Heterosexual sex	4 (9)	0 (0)	3 (20)	1 (5)
IDU and heterosexual sex	3 (6)	0 (0)	0 (0)	3 (16)
CDC HIV disease stage				
A	17	8	3	6
B	18	4	4	10
C	12	1	8	3
CD4 cell count, cells/μL	169 (82–371)	270 (105–515)	83 (22–179)	141 (82–295)
ΔRVSP-RAP, mm Hg	60 (50–75)	64 (52–78)	64 (52–75)	59 (49–75)
NYHA class	2.0 (2.0–2.0)	2.0 (1.5–2.0)	2.0 (2.0–2.0)	2.0 (2.0–2.0)

NOTE. Data are no. (%) of patients or median values (interquartile range), unless otherwise indicated. The 3 subgroups did not differ significantly in baseline characteristics (χ^2 or Kruskal-Wallis test, as appropriate), except for a later date of diagnosis in the HAART group. ART, antiretroviral therapy; CDC, US Centers for Disease Control and Prevention; IDU, injection drug use; MSM, men who have sex with men; NRTI, nucleoside analog reverse-transcriptase inhibitor; NYHA, New York Heart Association; ΔRVSP-RAP, elevation of right ventricular systolic pressure over right atrial pressure, measured by echocardiography.

[a] Data are no. of males/total no. of patients in the study (%).

the baseline date for patients receiving HAART was substantially later, and patients who did not receive any ART were all IDUs.

Incidence over time. The incidence of PAHRH was calculated as the percentage of the total number of SHCS participants in whom PAHRH was diagnosed during each year. The highest incidence numbers were observed between 1990 and 1995, with a maximum of 0.24% in 1993. From 1996, concurrently with the introduction of HAART, the incidence fell significantly to 0.06%–0.09% (table 2).

Treatment. The median follow-up duration was 2.7 years (range, 3 days to 10.5 years). The drugs used in the treatment of PAHRH included diuretics in 17 patients, calcium-channel blockers in 6 patients, oral anticoagulation agents in 13 patients, intravenous epoprostenol in 1 patient, and inhaled iloprost in 7 patients (a possible effect of iloprost therapy on the final echocardiography was present only for 4 patients; in the remaining patients, iloprost therapy was initiated only after the final echocardiography was performed). After the diagnosis of PAHRH, ART was prescribed to 34 patients. Among them, 15 patients were treated with NRTIs only, whereas 19 patients received HAART.

Hemodynamic data and effect of ART. One or several follow-up Doppler echocardiograms were available for 35 patients to document the continued course of PAHRH (table 3). When stratified by the anti-HIV treatment received between the first and final echocardiography, the 3 treatment groups had similar baseline RVSP-RAP gradients. The median change in the pressure gradient between the first and final echocardiography was +25 mm Hg in the 9 patients who did not receive ART, −3 mm Hg in the 12 patients who received NRTIs, and −21 mm Hg in the 14 patients who received HAART ($P <$ .005, by the Kruskal-Wallis test, with significant differences between the non-ART and NRTIs groups and the non-ART and HAART groups, by Dunn's multiple comparison test). The 3 groups also differed significantly in the proportion of patients in whom the pressure gradient increased or decreased by >10 mm Hg during the follow-up period. Because no relevant changes occurred in systemic blood pressure or heart rate, decreases in forward cardiac output could be excluded as a cause for the observed changes in the RVSP-RAP pressure gradient. The time course of the pressure gradient during the observation period is plotted in figure 1. Although the NYHA functional class values increased during the follow-up period for patients who were not receiving ART and for patients who were receiving NRTIs, patients receiving HAART showed stable to slightly decreasing values, illustrating clinical stabilization or improvement. In a multiple logistic regression analysis that was adjusted for age, sex, IDU, and AIDS stage at the time of

Table 2. Incidence of newly diagnosed cases of pulmonary hypertension (PH) per year as percentage of the total number of Swiss HIV Cohort Study (SHCS) participants with a follow-up visit during that year.

Year	New PH cases, no.	Active SHCS participants, no.	Yearly incidence of new PH diagnoses, %
1988	1	1433	0.07
1989	1	2109	0.05
1990	6	2832	0.21
1991	2	3246	0.06
1992	4	3263	0.12
1993	7	2953	0.24
1994	4	2751	0.15
1995	5	2910	0.17
1996	3	3344	0.09
1997	3	3929	0.08
1998	4	4440	0.09
1999	3	4839	0.06
2000	3	5203	0.06
2001	1	5432	0.02

NOTE. The trend showing decreasing incidence since 1995 was statistically significant ($P = .027$, by Poisson regression analysis).

PAHRH diagnosis, both overall duration of ART (OR, 1.79 per year of treatment; 95% CI, 1.11–2.90 per year of treatment; $P = .017$) and overall duration of HAART (OR, 1.94 per year of treatment, 95% CI, 1.17–3.25 per year of treatment; $P = .011$) were significant predictors of a pressure gradient decrease of >10 mm Hg.

Mortality. Thirty-one of 47 patients died during the follow-up period; 16 were still living at the end of the study. Median survival duration after the diagnosis of PAHRH was 2.7 years (range, 2 days to 10.5 years). Of the reported fatal cases, 10 deaths (32%) were attributable to the consequences of PAH (including right heart failure, cardiogenic shock, and sudden death), 16 deaths (52%) were attributable to HIV infection or other diagnoses, and 5 patients died of an unknown cause (16%). All 13 patients who were not treated with ART and all 15 patients who were treated with NRTIs only died, with an estimated median survival duration of 1.5 and 1.7 years, respectively (Kaplan-Meier estimates) (figure 2). In contrast, only 3 of 19 patients treated with HAART died. PAHRH was the direct cause of death in 5 (38%) of 13 of untreated patients, in 4 (27%) of 15 of those treated with nucleoside analogs, and in 1 (5%) of 19 of those treated with HAART ($P = .065$). In a multivariable Cox regression analysis that was adjusted for age, sex, IDU, and AIDS stage at the time of PAHRH diagnosis, HAART significantly decreased the risk of death (hazard ratio [HR], 0.075; 95% CI, 0.02–0.28; $P < .001$).

The mortality rate was 67% (24 of 36 patients) among individuals whose risk for HIV infection was IDU, which was comparable with the 64% mortality rate (7 of 11 patients) in the other risk groups. However, all untreated patients who died belonged to the IDU group, illustrating that ART is more difficult to initiate in this risk group.

The mortality rate among the 35 patients who underwent follow-up echocardiography was significantly lower in those who received HAART (table 3). In a multivariable Cox regression analysis that was adjusted for age, sex, IDU, and AIDS stage at the time of PAHRH diagnosis, both HAART (HR, 0.034; 95% CI, 0.005–0.23; $P < .001$) and a pressure gradient decrease of >10 mm Hg (HR, 0.97; 95% CI, 0.94–0.998; $P < .05$) significantly reduced the risk of dying.

DISCUSSION

Over a 13-year period, PAHRH was diagnosed in 47 patients. This represents a prevalence of 0.4% within the entire HIV-infected population in the SHCS (which involved 11,894 participants through the end of 2001) and an incidence of 0.02%–0.24% per year. The prevalence we observed is only slightly less than that observed in previous studies, in which the prevalence was estimated to be 0.5% [2, 3]. Although the number of newly diagnosed cases per year is relatively small, the decreasing incidence of PAHRH from 1996 through 2001 correlates well with the introduction of HAART and, in addition, indirectly supports the efficacy of HAART for treatment or prevention of PAH. This is in contrast to the retrospective study by Pugliese et al. [34], which found that PAH cases were more frequent among patients currently receiving HAART (2.0%) than among patients who received previous treatment with ART regimens (0.7%). However, Pugliese et al. [34] do not mention the number of untreated HIV-infected patients in their center, and the increased number of patients with PAHRH during receipt of HAART may simply reflect the recommendation known at that time that all patients with PAHRH should be treated with HAART.

In accordance with previous publications, the demographic data of out patients confirm that PAHRH occurs in all stages of HIV infection without an obvious relation to immune deficiency. Nineteen of our patients (40%) had CD4 lymphocyte counts of >200 cells/μL, which corresponds to the ratio in other reports [1, 24]. IDU and male-male sex predominated among our patients, but PAHRH occurs in all risk groups. Among our patients, there was a small predominance of men over women.

In 35 patients (74%), a minimum of 1 follow-up Doppler echocardiogram was available, allowing us to determine the course of the pressure gradient over time. Doppler echocardiography is recognized as a useful and reliable tool for documenting the time course of PAH [35]. Because shifts in the pressure gradient occurred without concurrent changes in sys-

Table 3. Time course in 35 patients with echocardiographic follow-up data.

Characteristic	Treatment group		
	No ART during follow-up (n = 9)	Only NRTIs during follow-up (n = 12)	HAART during follow-up (n = 14)
Follow-up duration, years	2.4 (0.8–5.3)	1.7 (1.1–3.1)	4.8 (2.5–8.5)
Duration of ART after PH diagnosis, years	…	0.9 (0.3–1.5)	3.6 (1.9–6.9)
Duration of HAART after PH diagnosis, years	…	…	3.4 (1.9–5.6)
CD4 cell count at baseline, cells/μL[a]	300 (115–515)	82 (26–155)	240 (95–396)[c]
Final known CD4 cell count, cells/μL[b]	120 (68–385)	39 (9–189)	373 (163–510)[d]
Final known HIV-1 RNA load <400 copies/mL	1/4 (25)	0/3 (0)	9/12 (75)
CDC HIV disease stage			
Stage B/C	2 (22)	1 (8)	1 (7)
Stage C	2 (22)	1 (8)	1 (7)
Cause of death			
Cardiac reasons	5 (56)	4 (33)	0 (0)
HIV-related or other diagnoses	3 (33)	5 (42)	2 (14)[f]
Unknown	1 (11)	3 (25)	0 (0)
Total	9 (100)	12 (100)	2 (14)
Time from first to last echocardiography, years	1.4 (0.4–4.1)	1.0 (0.3–1.8)	3.6 (1.9–8.1)
No. of echocardiograms per patient	3.0 (2.0–5.0)	2.5 (2.0–4.0)	3.0 (2.0–6.5)
ΔRVSP-RAP, mm Hg			
First	60 (47–84)	62 (54–76)	60 (51–83)
Final	93 (64–98)	58 (49–66)	50 (36–61)
Change	+25 (+6 to +35)	−3 (−24 to +4)	−21 (−30 to −6)[d]
Increase by >10 mm Hg	6 (67)	2 (17)	2 (14)[e]
Decrease by >10 mm Hg	0 (0)	5 (42)	10 (71)[f]
NYHA class			
Time of diagnosis	2.0 (1.0–2.5)	2.0 (2.0–2.0)	2.0 (2.0–2.0)
Change	+1.0 (+1.0 to + 2.0)	+1.0 (−0.5 to +2.0)	0 (−1.0 to 0)[c]
Treatment			
Diuretics	4 (44)	5 (42)	6 (43)
Digoxin	1 (11)	3 (25)	2 (14)
Calcium-channel blockers	2 (22)	2 (17)	1 (7)
Vasodilators other than calcium channel blockers (iv epoprostenol, inhaled iloprost)	0 (0)	1 (8)	3 (21)

NOTE. Data are no. (%) of patients or median values (interquartile range). ART, antiretroviral therapy; CDC, US Centers for Disease Control and Prevention; IDU, injection drug use; NRTI, nucleoside analog reverse-transcriptase inhibitor; NYHA, New York Heart Association; PH, pulmonary hypertension; ΔRVSP-RAP, elevation of right systolic ventricular pressure over right atrial pressure.

[a] n = 9, 12, and 13 for each column, respectively.
[b] n = 8, 10, and 14 for each column, respectively.
[c] $P < .05$, by the Kruskal-Wallis test for comparisons between the 3 groups.
[d] $P < .005$, by the Kruskal-Wallis test for comparisons between the 3 groups.
[e] $P < .02$, by the χ^2 test for comparisons between the 3 groups.
[f] $P < .005$, by the χ^2 test for comparisons between the 3 groups.

temic blood pressure or heart rate, a decrease in forward cardiac output could be excluded as a cause for the observed changes in the RVSP-RAP pressure gradient.

In our patient population, HAART and, to a lesser degree, treatment with NRTIs resulted in a significant beneficial effect on the course of the pressure gradient, and HAART led to a decreased or stabilized NYHA functional class value. In the group of 35 patients for whom follow-up Doppler echocardiograms were available, the RVSP-RAP gradient increased by a median of 25 mm Hg in 9 patients who were not treated with ART, decreased by a median of 3 mm Hg in 12 patients who were treated with NRTIs, and decreased by a median of 21 mm Hg in 14 patients who received HAART ($P < .005$).

It must be emphasized, however, that the treatment groups—

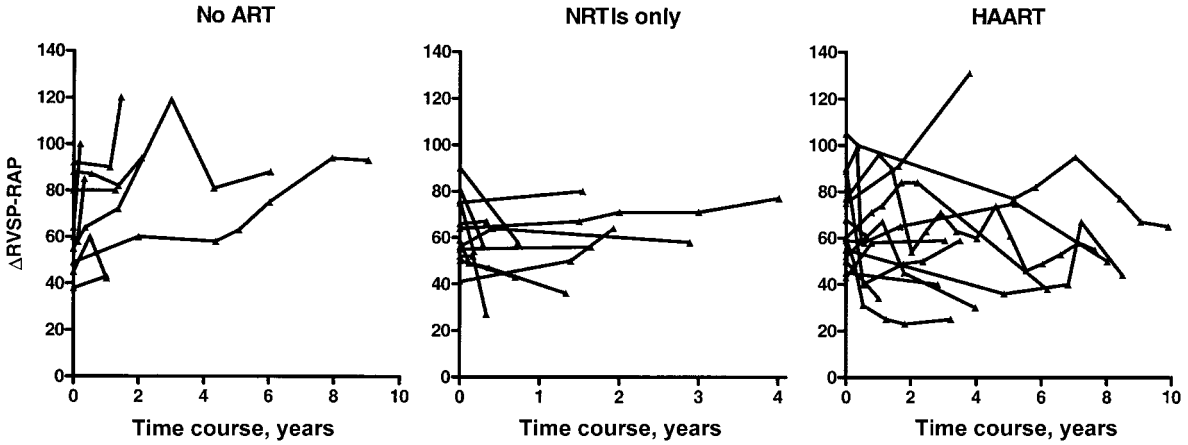

Figure 1. Time course of the elevation of right ventricular systolic pressure over right atrial pressure (ΔRVSP-RAP), assessed by continuous-wave Doppler echocardiography, in patients receiving no antiretroviral therapy (ART) *(left),* compared with patients receiving nucleoside analog reverse-transcriptase inhibitors (NRTIs) only *(middle)* and patients receiving HAART *(right).* The x axis of the middle graph is expanded because of the shorter follow-up period for these patients.

in particular, the HAART group—are not fully comparable and represent periods with different standards of care and means of clinical assessment. In many patients treated with HAART, the virus load was undetectable, but HIV RNA PCR was not available before the era of HAART. The possible correlation between suppressed virus load and echocardiogram findings could not be evaluated, because these assessments were frequently performed at different time points. Other baseline parameters may have had prognostic influence. The multiple logistic regression analysis and multivariable Cox regression analysis have therefore been adjusted for age, sex, IDU, and AIDS at baseline. None of these parameters showed any significant predictive effect on the hemodynamic response or survival, either in the univariable or in the multivariable analysis.

Seven patients treated with ART also received intermittent therapy with inhaled iloprost. During the 2 months of intravenous treatment, 1 of these 7 patients also received epoprostenol. In 3 of these 7 patients, all Doppler echocardiographic studies were performed without iloprost or epoprostenol treatment, and thus the results were not influenced by these agents. In 4 patients, ART was administered concomitant with inhaled iloprost therapy. In these 4 patients, the pressure gradient decreased, which was likely influenced by the concomitant treatment with inhaled iloprost. When these 4 patients were removed from the analysis, the median change in the pressure gradient was +1 mm Hg in 11 patients treated with NRTIs (not including data for 1 patient) and −19 mm Hg in patients receiving HAART (not including data for 3 patients), indicating that concomitant treatment with iloprost and epoprostenol did not change the overall results in any major way. None of the patients were treated with bosentan.

The median survival duration was similar between untreated patients and those who received NRTIs but was significantly improved among those who received HAART. This is not surprising, according to the known benefit of combination ART [36, 37]. Because HAART decreased mortality directly attributable to PAH as well as mortality due to HIV infection and other causes, the reduction in the right ventricular workload seems to have directly translated into a clinical benefit.

Even if HIV or its proteins have not been identified in the pulmonary vascular smooth muscle or endothelium in patients with PAHRH [10], HIV infection induces a chronic inflammatory state and persisting immune activation [38]. Instead, it is hypothesized that HIV-infected macrophages may release abnormal types and/or quantities of cytokines that sequentially

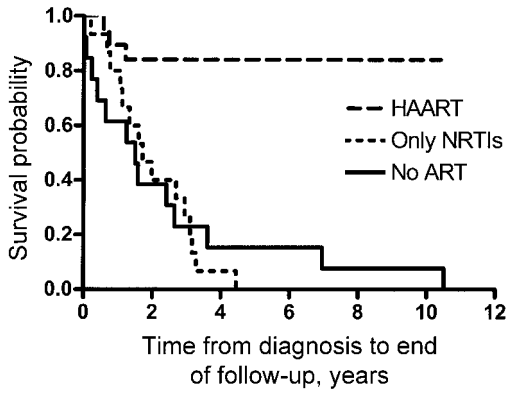

Figure 2. Probability of survival from the time of diagnosis of pulmonary hypertension until the end of the follow-up period, stratified by the type of antiretroviral therapy received ($P < .0001$, by the log-rank test). ART, antiretroviral therapy; NRTI, nucleoside analog reverse-transcriptase inhibitor.

lead to enhanced leukocyte adherence, growth factor secretion, and endothelial proliferation [4]. In fact, in several studies, high levels of endothelin-1, IL-1a, IL-6, and platelet-derived growth factor in patients with PAHRH have been found [11, 12, 39]. HAART downregulates viral replication and leads to a decrease in abnormal rates and/or types of T cell activation [40]. It is conceivable that reduction of the chronic stimulation of the immune system brought about by ART might also decrease the (still unknown) driving force behind the development of PPH. Recently, human herpesvirus 8 (HHV-8) has been suggested as a possible etiologic agent in PPH [41], but no data exist on the association between HHV-8 and PAHRH. Because HAART itself decreases the incidence of Kaposi sarcoma (which is associated with HHV-8) and decreases circulating HHV-8 viremia [20, 42], further study regarding the possible association between HHV-8 and PAHRH seems to be warranted.

In conclusion, an indirect pathogenic relationship between HIV infection and the incidence of PAH is strongly suggested in the face of the improved hemodynamic parameters in our patients who received ART, particularly HAART. This study confirms the recommendation in a previous study [24] to treat all patients with PAHRH with HAART, irrespective of their CD4 lymphocyte counts.

STUDY GROUP MEMBERS

The members of the Swiss HIV Cohort Study (SHCS) are S. Bachmann, M. Battegay, E. Bernasconi, H. Bucher, Ph. Bürgisser, S. Cattacin, M. Egger, P. Erb, W. Fierz, M. Fischer, M. Flepp, A. Fontana, P. Francioli (President of the SHCS), H. J. Furrer (Chairman of the Clinical and Laboratory Committee), M. Gorgievski, H. Günthard, B. Hirschel, L. Kaiser, C. Kind, Th. Klimkait, B. Ledergerber, U. Lauper, M. Opravil, F. Paccaud, G. Pantaleo, L. Perrin, J.-C. Piffaretti, M. Rickenbach (Head of Data Center), C. Rudin (Chairman of the Mother and Child Substudy), J. Schüpbach, R. Speck, A. Telenti, A. Trkola, P. Vernazza (Chairman of the Scientific Board), R. Weber, and S. Yerly.

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